

Sentinel Symptoms of Climate Change

Indicators for Related Health Effects

Greenhouse gas emissions are widely acknowledged to be contributing to climate change–related health effects that vary by location, and are expected to continue doing so for many years, even if substantial emission cuts occur. A workgroup of the Council of State and Territorial Epidemiologists has identified a set of indicators that it says will allow national and local officials in the United States to better predict any such changes and consequences and to take appropriate action as it becomes warranted [*EHP* 117:1673–1681; English et al.]. The team also identified the data needed for tracking these indicators and ascertained whether the data exist, must be improved, or must be generated. They say this is the first effort to synthesize and evaluate related information published by many sources.

The team determined the best indicators of environmental changes due to climate change are quantity of greenhouse gas emissions, air quality (in particular ozone), air mass stagnation events (such as those caused by temperature inversions), temperature and humidity, pollen loads, ragweed occurrence, drought incidence, drinking water scarcity, and occurrence of wildfires and harmful algal blooms. Data for some of these indicators are strong and/or expected to improve soon, as in the case of greenhouse gases, temperature, air mass stagnation events, and drought. Data on other indicators, such as pollen, harmful algal blooms, and ozone, require substantial improvement.

For indicators of human death and illness, the authors recommend tracking

excess numbers of each that can be attributed to events related to climate change. Doing so will require significant improvements in existing data and methods, such as more comprehensive reporting of emergency room visits and hospitalizations related to heat waves, floods, and other extreme weather events. For infectious diseases, the targeted culprits are West Nile virus, Lyme disease, dengue fever, coccidioidomycosis, and hantavirus cardiopulmonary syndrome.

The authors note that some segments of the population may be especially vulnerable to certain effects of climate change. These groups include children; the elderly; pregnant and nursing women; those with disabilities and preexisting conditions such as asthma, chronic obstructive pulmonary disease, and obesity; people living in poverty or social isolation or without access to transportation; and those living within 5 km of a coast that is highly vulnerable to sea level rise, or in a 100- or 500-year flood zone. Awareness of these vulnerable subpopulations will be important in planning appropriate prevention and intervention activities.

Data for indicators of adaptability are sparse because most efforts so far have focused on mitigating climate change, not adapting to it. The authors propose that such indicators might include access to public cooling centers during heat waves, the existence of early warning systems for heat waves, mitigation plans to reduce urban heat islands, the number and quality of surveillance systems available to collect data on climate–health effects, and the availability of local public health workers and task forces trained in climate change research, surveillance, and adaptation.

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Micro Management

Understanding How Diesel Exhaust Particles Alter Cellular Processes

Scientists have known for decades that people living in cities are more susceptible to certain respiratory diseases than are their countryside counterparts. But they haven't been able to explain why one urbanite develops severe asthma while his neighbor breathing the same city air has healthy lungs. Now researchers are beginning to solve that riddle as they delve into epigenetics, the emerging science of how environmental factors alter gene expression [*EHP* 117:1745–1751; Jardim et al.].

This new study focuses on airborne particulate matter, which has long been linked with respiratory disease. Emissions from diesel engines are a prominent source of particulate matter, and diesel exhaust particles (DEP) are classified by the U.S. Environmental Protection Agency as a likely carcinogen. DEP also has been associated with several respiratory disorders including pulmonary inflammation, asthma, and chronic obstructive pulmonary disease.

The authors speculated that pulmonary inflammation due to DEP exposure could be the result of altered microRNA (miRNA) expression, or activation, in cells lining the respiratory tract. MiRNAs are small molecules that regulate gene expression. Studies have connected aberrant miRNA expression with several diseases including cancer, heart disease, neurodegenerative disorders, and congenital

organ defects. Few studies, though, have examined whether exposures to environmental contaminants alter miRNA expression.

The authors studied miRNA expression in bronchial epithelial cells, one of the first targets of inhaled particulates. They collected the cells from the airways of healthy, nonsmoking adults and cultured them in a novel air–liquid interface in which differentiated cells were exposed on one side to air, mimicking the environment of the human airway. DEP generated by a diesel automobile engine was suspended in a liquid that was poured on the cell culture.

MiRNA expression changed significantly following DEP exposure. Expression increased in many of the miRNAs and decreased in others. Software identified interrelations between the expression of different miRNAs to assess whether the pattern of up- and down-regulation was consistent with specific biologic pathways. The authors report that pathways involved in inflammation and tumorigenicity are implicated by the patterns they observed.

Earlier studies have shown that DEP prompts the release of several proinflammatory immunoregulatory proteins called cytokines. The authors of the current study suggest this response may be at least partly regulated by changes in the pattern of miRNA expression. The authors believe these alterations may be the first steps toward respiratory disease, and they predict future studies will provide a clearer picture of how expression patterns relate to disease.

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Delivering New Data

Local Traffic Pollution and Pregnancy Outcomes

Up to 35% of preterm births are due to preeclampsia, a complication in 2–8% of pregnancies that is characterized by maternal high blood pressure, edema, protein in the urine, and abnormal liver function. Exposure to certain air pollutants is associated with prematurity and may also be linked with preeclampsia. A new study is the first to home in on specific components of air pollution—those generated by traffic—as being associated with preeclampsia and further supports their role in preterm birth [EHP 117:1773–1779; Wu et al.].

Preeclampsia, which resolves only with delivery of the baby, can cause maternal illness and death, intrauterine growth restriction, preterm birth, and infant death. Each year more than half a million infants in the United States are born prematurely (at less than 37 weeks' gestation) and consequently face increased risks for developmental delays, lifelong health problems, and neonatal death. These challenges are particularly severe for infants born prior to 30 weeks' gestation.

The study was based upon 81,186 singleton births that occurred during 1997–2006 at four Southern California hospitals within the same health care system. The system's database provided information on the mothers' demographic characteristics, medical history, and pregnancy complications; their home address at the time they gave birth; and their infants' gestational age, sex, and birth weight.

Traffic pollution generated within a 3-km radius of each mother's residence was estimated using a comprehensive dispersion model that

incorporated meteorologic variables (such as atmospheric stability and wind), roadway geometry, traffic counts, and vehicle emission factors. The exhaust components nitrogen oxides (NO_x) and particulate matter smaller than $2.5 \mu\text{m}$ ($\text{PM}_{2.5}$) served as surrogates for local traffic pollution in the model.

The researchers estimated average exposures over the entire pregnancy at approximately 7 ppb for NO_x and $2 \mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$. After accounting for other factors that might be related to preeclampsia and exposure, the authors estimated that pregnant women in the highest quartile of $\text{PM}_{2.5}$ exposure had a 42% increased relative risk of preeclampsia compared with women in the lowest quartile, and those in the highest quartiles of NO_x and $\text{PM}_{2.5}$ exposure had 128% and 81% higher relative risk than women in the lowest quartiles, respectively, for delivery at less than 30 weeks' gestation. The sophisticated dispersion model and detailed individual clinical data are particular strengths of the study, but the findings are limited by information that was not available, such as workplace exposures, changes in residence during pregnancy, and maternal smoking.

The researchers speculate that the toxic mechanisms described in air pollution studies of respiratory and cardiovascular diseases—specifically, oxidative stress and a generalized inflammatory response—might also partly explain preterm delivery and preeclampsia. They emphasize that the current study does not specifically indict NO_x and $\text{PM}_{2.5}$, although the results support a connection between traffic-related air pollution and adverse reproductive outcomes.

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You Are Where You Live

The Interrelationship of Air Pollution, Address, and Walkability

Exposure to air pollution adversely affects human health by triggering or exacerbating a number of conditions such as asthma and heart disease. Likewise, physical inactivity has been linked to negative health consequences including heart disease and diabetes. Now for the first time a new study offers a quantitative analysis of the intersection between neighborhood “walkability”—or how conducive the neighborhood is to walking—and exposure to air pollutants [EHP 117:1752–1759; Marshall et al.].

The authors analyzed concentration estimates of nitric oxide (NO), a marker of fresh vehicle exhaust, and ozone (O_3), a secondary pollutant formed in the atmosphere from vehicle emissions and other pollutants. Concentrations were estimated for the months of May–September. The authors then compared those levels against neighborhood walkability scores, which they calculated for 89% of the postal codes in Vancouver, British Columbia (the average postal code for the city comprises 39 people or 0.05 km^2). Walkability scores were calculated based on residential density, intersection density, retail floor-area, and land-use mix of the postal code area. The study did not measure people's daily exercise levels or their exposure to

air pollution—both of which may vary within a neighborhood and even within a single household.

The authors report that lower-income areas tended to have higher walkability scores and lower O_3 concentrations than did higher-income areas, but had higher NO concentrations. That finding reflects the tendency of lower-income areas to fall in busier urban areas whereas middle-income areas tend to fall farther from the city center. “Sweet spot” neighborhoods with high walkability and low pollution tended to be located near but not at the city center.

They typically featured highly connected streets, mixed land uses, sidewalks, and an absence of large parking lots; they also tended to be in higher-income areas. “Sour spot” neighborhoods with high pollution and low walkability tended to be located far from the city center.

The findings indicate that walkable urban settings can offer health benefits but may also come with health costs when exposure to air pollution is considered. The authors write that high NO exposure may occur in low-income areas and in areas where walking, biking, and other forms of “active transportation” are encouraged, and that strategies are required to mitigate exposure to high concentrations of air pollutants. This type of analysis could be used to monitor changes over time in future urban development and redevelopment projects.

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Vancouver, British Columbia